VOLUME XVI NOVEMBER 1960 NUMBER 11



# Clinical Proceedings

## OF THE CHILDREN'S HOSPITAL

WASHINGTON, D. C.

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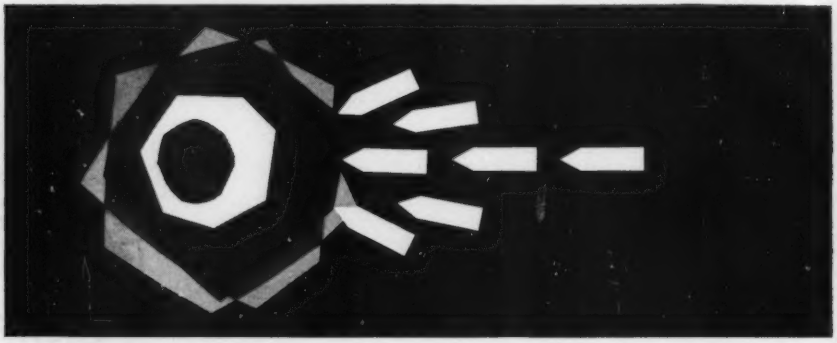

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**references:** (1) Clinical Research Division, Mead Johnson & Company. (2) Howell, C. M., Jr.: Evaluation of Methdilazine Hydrochloride as an Antipruritic Agent, North Carolina M. J. *21* (May) 1960 (in press). (3) Wahner, H. W., and Peters, C. A.: An Evaluation of Some Newer Antihistaminic Drugs Against Pollinosis, Proc. Staff Meet. Mayo Clin. *35*:161-169 (March 30) 1960.

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17740

# CLINICAL PROCEEDINGS
## OF THE CHILDREN'S HOSPITAL
2125 13th Street, N.W., Washington 9, D. C.





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# Panel Discussion: Cryptorchidism

JOHN A. WASHINGTON, M.D.*
NEVILLE K. CONNOLLY, M.D.†
HILBERT S. SABIN, M.D.†

Dr. Washington:

A panel similar to this several years ago reached the conclusion that, with the evidence at hand, the proper handling of children with cryptorchidism called for operating at the age of 6 or 7 years. We have largely adhered to that rule at Children's Hospital. At that time one stubborn dissenter to early operation was Dr. Lawson Wilkins in Baltimore. I talked to Dr. Wilkins the other night and he says that he is still unconvinced that operating early is justifiable. He is impressed by the number of operated cases that show atrophy and by the number of unoperated cases in which the testes come down undamaged spontaneously at puberty. The testes are small, he says, when they come down, but they enlarge and become normal in a short time. In a February 1957 issue of the Journal of the American Medical Association there were two articles with diametrically opposed views on the handling of cryptorchidism. One was by Kimbrough and Reed[1] at Walter Reed Army Hospital, who advocated operating on these boys when they were no older than 6 and as young as 3; the other was by Drake[2] in Minnesota, who advocated never operating before the age of 16 or 17. Kimbrough and Reed's reasoning was based mainly on histologic studies of testes biopsied at operation which indicated that the longer they remain out of the scrotum the more they are damaged. Drake, who is opposed to operating except as a last resort, attempted to compare postadolescent fertility in two series. For his surgical series, he chose that of Gross of Boston, which he thought presented the best surgical results reported and was large enough to include a significant number of bilateral cases. Of course, the ultimate fertility of bilateral cases supplies the proof of the pudding. Of some 1,200 cases, Gross was able to do fertility follow-ups on 38 bilateral cases and found acceptable fertility in 79 per cent.[3] Drake picked for comparison an unoperated series reported by Johnson in New York in 1939.[4] Johnson worked at a boys' club and performed repeated

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examinations on large numbers of boys from 7 to 17 years of age; some of his observations are summarized below:

*Age of Descent of Testes*

| Age* | Bilateral | Unilateral | Total |
|---|---|---|---|
| 7 | 6 | 3 | 9 |
| 8 | 9 | 3 | 12 |
| 9 | 14 | 10 | 24 |
| 10 | 12 | 11 | 23 |
| 11 | 28 | 23 | 51 |
| 12 | 36 | 34 | 70 |
| 13 | 28 | 25 | 53 |
| 14 | 20 | 12 | 32 |
| 15 | 7 | 12 | 19 |
| 16 | 1 | 4 | 5 |
| 17 | 1 | 1 | 2 |
| — | — | — | — |
| Total | 162 | 138 | 300 |

* Age at which spontaneous descent occurred.

He thinks that operating on undescended testes is being overdone because in this total of 300 cases, only 2 boys had testes which failed to enter the scrotum by age 17. The greatest incidence of descent occurred between the ages of 11 and 13. He *assumed* that these spontaneously descending testes were fertile because they appeared normal when palpated.

It would seem that there is a spectrum of opinions to choose from: at the one extreme stand Kimbrough and Reed who advocate operating when the child is 3 to 6 years of age; and at the other extreme, Johnson and Drake who advocate operating as a last resort. In between are Gross, who operates between 9 and 11 years, and Wilkins, who waits until puberty is well under way and then operates if he feels that puberty is not going to do the job.

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Dr. Connolly:

There is obviously much divergence of opinion when to operate on undescended testes for the reason that it is very difficult to decide which cases are satisfactory for expectant treatment. There are different criteria for treating, and there is no uniformity of feeling as to the goal to be achieved. It is necessary to consider first the objective of treatment. This

should be to achieve normal fertility in all patients. Since a unilateral cryptorchid is usually fertile, the only ones whose follow-up will elucidate the spermatogenic function of the undescended testes are those with bilateral undescended testes. An adequate sized normal-feeling testis at the bottom of the scrotum does not insure normal spermatogenesis.

Normal spermatogenesis is difficult to estimate. Testicular biopsy is the ideal, but these biopsies are not readily obtained, as patients seem to be reluctant to submit themselves to such a procedure. Sperm counts may be significant but only in the bilateral cases. It is really in bilateral cases where the family history has been followed over a number of years that we can obtain the best idea of our results. Even in those cases where the results of bilateral cases have been followed and the ultimate fertility shown, no mention has been made as to starting condition nor have biopsies been taken at the time of operation.

There is also the cosmetic and, allied to that, the psychological reason for treating these cases. Undoubtedly it is considered a normal male appearance to have two gonads in the scrotum, and a lack of one of these can have a considerable psychological effect on the individual which is difficult to overestimate. This is particularly true of the young boy going away to school or appearing in the locker room for the first time. In unilateral cases, besides the psychological reasons, it seems wise to bring the second testis down in order to increase the spermatogenesis so that the individual may have a spare or, as one might say, an insurance policy.

In order to advise satisfactorily on the ideal method of treatment in any case, it is imperative to keep in mind that cryptorchidism is not one condition but embraces at least five different conditions. The first is that of a generalized glandular deficiency, such as hypopituitarism, where the bilateral cryptorchidism is merely one feature of a symptom complex. These cases are not suitable simply for orchidopexy but must be treated for their whole deficiency and might respond well to hormone therapy. The second class includes those conditions in which there is primary testicular dysplasia. In these cases it is not the descent of the testis which is at fault but the histological pattern of the testis itself. Unfortunately it is not possible to diagnose this condition of primary testicular dysplasia without a biopsy of the gonad, and in fact these cases are not diagnosed unless a biopsy is taken at the time of operation. The remaining three types depend on the site of the testis at the time when the patient is examined. It seems that this may well be of great importance both with regard to prognosis and treatment. The testis may be on the normal line of descent as illustrated in figure 1.* If the testis is abdominal or, as shown, entrant inguinal, it will not be able to be palpated. If it is emergent in-

* Figures 1–4 reprinted from Louw, J. H.: Undescended testis, published in the South African Medical Journal, volume 28, 18 September 1954, pages 807–812.

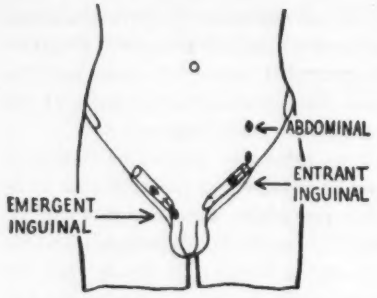


FIG. 1. Normal line of descent of testes

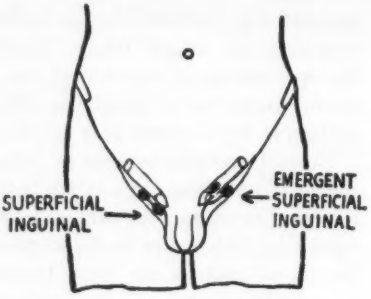


FIG. 2. Inguinal ectopic testes

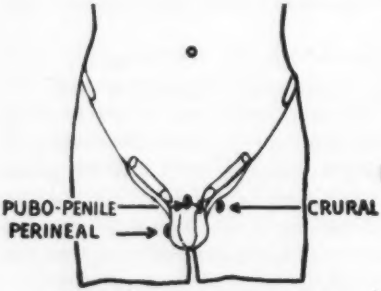


FIG. 3. Other types of ectopia

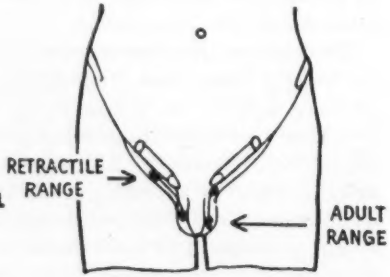


FIG. 4. Normal varieties

guinal, it will not be able to be palpated while it is in the inguinal canal, but on careful examination can be forced out of the inguinal canal and will be felt to proceed down towards the upper part of the scrotum. This group of undescended testes would best be called incompletely descended in contrast to the second group shown in figures 2 and 3.

In the second group, in all but the emergent superficial inguinal position, the testis has descended but is ectopic in position. In the example of the superficial inguinal testis (fig. 2), the testis is clearly palpable under the skin over the inguinal canal; it is not in the inguinal canal and this can be demonstrated clearly at operation. If it is in the inguinal canal, it will be covered by the fascia of the external oblique and will not be palpable as a distinct organ, although a vague sense of a mass may be felt, or possibly the patient may feel typical testicular pain when it is pressed upon. The emergent superficial inguinal testis in figure 2 must not be confused with the emergent inguinal in figure 1; the difference here is that when the testis is forced out of the canal with lateromedial milking, it will be felt to turn laterally as it comes out of the external ring and become palpable in this

position, a little below and lateral to the external ring. This is an incompletely descended ectopic testis. Figure 3 merely illustrates certain of the other ectopic positions in which a testis can be found. This group of ectopic testes have all descended but are in the wrong position. There is no possible treatment for any of these except surgical reposition in the correct place. In the majority of cases this is a reasonably easy operation, since the blood vessels and the vas are both long enough to allow the testis to be put at the bottom of the scrotum without difficulty.

The final group which must be mentioned is shown in figure 4; these are the normal varieties. Appreciation of this point is vital in the assessment of the results of treatment. In the normal adult range of the testis it will be seen that the testis remains within the scrotum although it may be pushed right up to the top. In the retractile range it will be seen that the testis can be pushed well up over the pubis and indeed over the inguinal canal. It is these cases which cause most of the confusion, as they can readily be confused with the emergent inguinal cases shown in figure 1, particularly if the retractile range in the infant does not allow the testis to come down beyond the upper part of the scrotum. However, it is considered that these are normal cases and if left alone the testis will descend normally. My own feeling is that the difficult case to decide about is

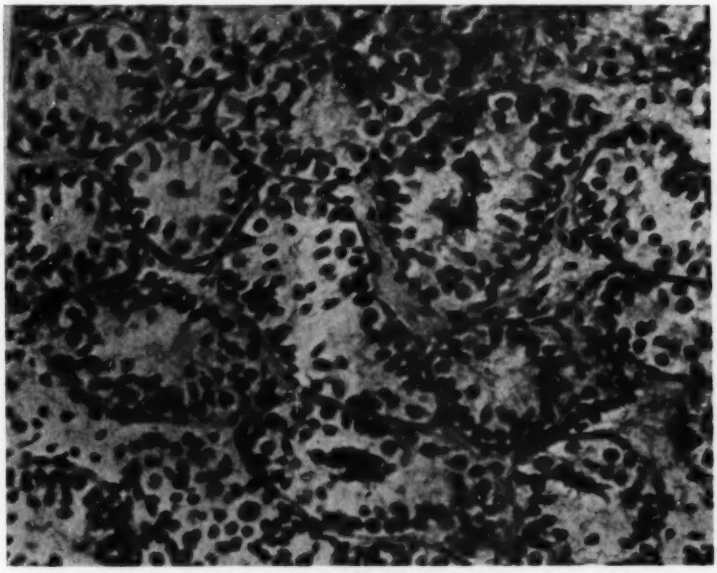

FIG. 5. Normal testis at age 4

the emergent inguinal, as in such a case, according to many observers, the testis will descend either at puberty or with hormone treatment. However, in my own mind I am not clear that this is true, and I believe that most of those that descend really belong to the retractile group.

It is interesting to consider Johnson's figures in the light of this classification. If Johnson's figures are true, they show that he did not have a single ectopic testis in the whole of his series, and yet in a number of surgical series it has been shown that the majority of cases are ectopic. Denis Browne[1] states that 80 per cent of his cases are ectopic, and Louw[2] found over 50 per cent of ectopic position in his cases collected in South Africa. It is equally difficult to believe that somebody with Johnson's experience should mistake retractile testes for undescended testes. However, there is something very unusual about his figures, and it is interesting to note that the time of maximum descent in his series is identical with the time at which the advocates of hormone therapy point out that they achieve their best results.

The age factor in treatment is another point which requires careful consideration. Here again I think the type of undescended testis that is being dealt with is important. If the testis is ectopic there seems to be no point whatsoever in waiting, either to see what will happen or for any

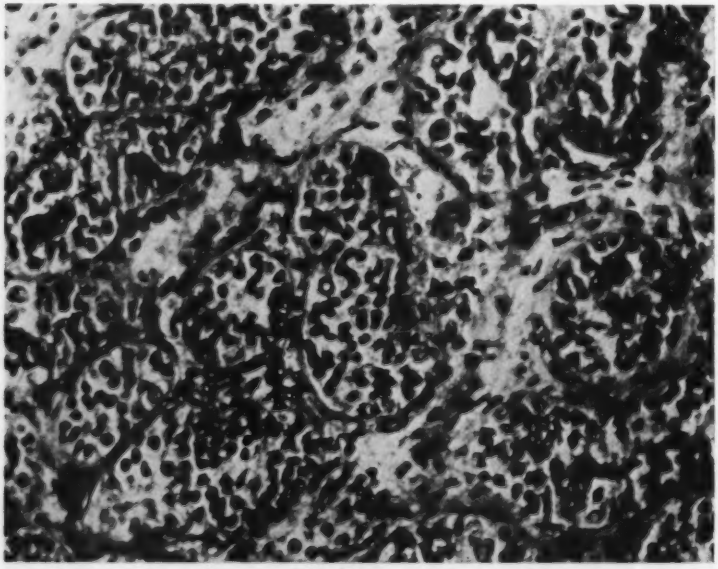

FIG. 6. Undescended testis, age 11, at orchidopexy

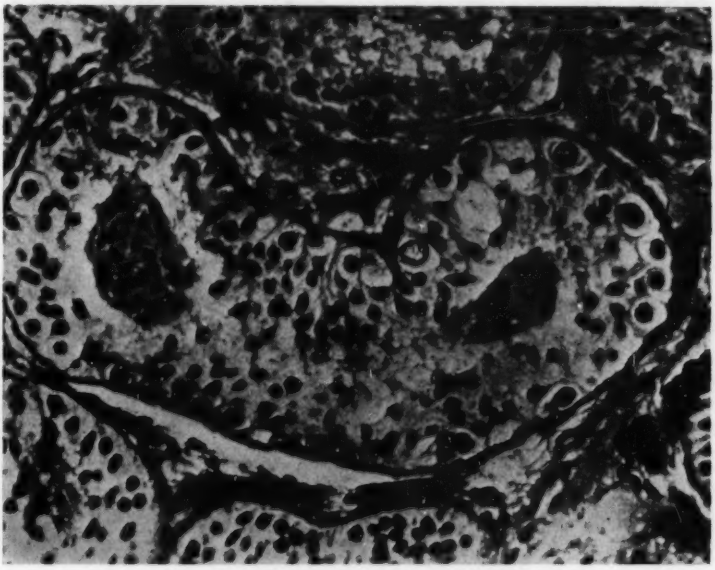

FIG. 7. Normal testis, age 12, prepubertal

other reason. As soon as such a diagnosis can be made satisfactorily, the patient may be operated on; one must remember that the longer the testis remains anywhere but in the scrotum the worse would seem to be the histology. There seems to be accumulating evidence that the histologic picture of the testis varies very considerably with age when it is not in the scrotum. Figures 5–8* illustrate vividly the change in appearance seen between descended and undescended testes. Accumulated work seems to show that this change becomes quite obvious from 5 or 6 years on and really marked after the age of 9. For this reason I feel that 5 or 6 years is the ideal time at which to operate in these cases of incomplete descent where the testis is in the right line but has not come far enough. The reason that one cannot be dogmatic about this point is that there is no evidence as to how much the testis can improve after it has been put in the scrotum. For instance, if a testis such as that shown in figure 8 is placed in the scrotum without damage to its blood supply, will the germinal epithelium regenerate and spermatogenesis occur? There is only one article that I know of in the literature which suggests this may occur,[3] and it would be interesting to have more evidence on this point. Probably when we have this

* Figures 5–8 reprinted from Charny and Wolgin: Cryptorchism, New York, Paul B. Hoeber, Inc., 1957.

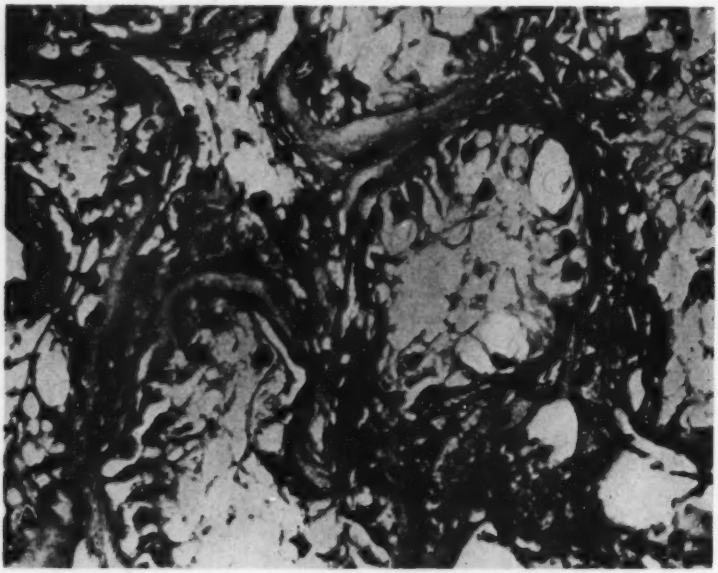

FIG. 8. Undescended testis, age 14, at orchidopexy

evidence we will find that to some extent improvement can take place but that there is a point of histological damage beyond which there will be no improvement.

There does not seem to be any place for hormone therapy in my estimation. Two types of hormones are used, chorionic gonadotropin and androgens. Chorionic gonadotropins cause an artificial onset of puberty first manifested by histological changes in the testes; consequently if the testis descends with hormone therapy it is exactly the same for the testis as waiting until puberty and allowing it to come down at that time. If waiting until puberty for the testis to descend is bad because of the potential histologic damage, giving hormones to bring on this puberty early to make it come down is just as bad. Studies have shown that after chorionic gonadotropin the testis may be just as deformed histologically as if it had been allowed to remain elsewhere than in the scrotum.[4] The androgens are even more dangerous. They may cause premature fusion of the epiphyses and should never even be attempted. Psychologically of course, giving injections to a child three times a week to get in the required 200,000 international units of chorionic gonadotropin over a period of 4 or 5 weeks is worse, to my mind, than operating on him. The hormones have been advocated because they will "make everything a bit bigger and the surgery

easier." This is false reasoning because as the size of the testis increases its demand for blood supply increases. Operating on a testis, no matter how carefully it is done, traumatizes its blood supply to some extent. It is therefore preferable to have the testis as inactive as possible during the time of the operation.

With regard to the operation, it is worth pointing out that a surprising number of people still perform the Torek operation and that this contravenes almost every principle of plastic surgery. The Torek procedure involves freeing up the testis, pulling it down through the scrotum and attaching it to the thigh. It is common knowledge that any tension on a pedicle will cause the end of the pedicle to slough. This is surely what the Torek operation must do. Even putting elastic band tension on the testis as is recommended by Gross[5] can be dangerous for this same reason. It seems far more satisfactory to place the testis as far down as it will comfortably lie without tension and without any pulling on the organ to stretch the blood supply. Not all testes can be placed in the scrotum with such an operation, but all testes should survive this operation and can be attacked again at a later date. After a year or so it is sometimes possible to get the testis the remainder of the way to the bottom of the scrotum. This is an additional reason for starting early, i.e., 5 or 6 years, and not waiting until the last possible moment to avoid histological change.

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Dr. Sabin:

I agree with almost everything you have said, Dr. Connolly. There is one controversial point in cryptorchidism which has not been covered so far, and that is the alleged increased occurrence of testicular tumors in cryptorchids. Almost every treatise on this subject that one reads says that testicular tumors occur from 10 to 20 times as frequently in undescended testicles as they do in scrotal testicles. That has been very interesting to me because, in 20 years of practice, I have seen many testicular tumors but have seen only one in an undescended testicle, and that was in an individual over 60 years old. It is therefore difficult to believe in the validity of such statistics.

As to the best time for bringing the testes down surgically, I personally believe that the preschool age is ideal. You can determine fairly well preoperatively whether the testicle is going to descend spontaneously or not. First of all, a testicle in the inguinal canal is not palpable; it is only the testicle that has emerged from the external inguinal ring that one can feel. If these palpable testes are in the normal line of descent and are freely movable, they will come down spontaneously; if they are ectopic, only surgery will get them in the right place. Frequently these ectopic testes have partially descended and then have moved around over the aponeurosis of the external oblique muscle; it is an extremely simple matter to bring these down; traction of any kind is unnecessary since the cord is perfectly adequate, and these testicles lie in the scrotum without being under any tension. Incidentally, the Walter Reed group has done further work which is thus far unpublished regarding the fertility of children whom they have operated on between the ages of 5 and 7, and they claim rather excellent results. A problem that arises in operating on unpalpable testicles is inability to find the testicle, or finding it at such a level that it cannot be brought down into the scrotum. I personally believe that such a testicle should be sacrificed and removed at the time of operation. It will undoubtedly have no spermatogenic activity, and there is *a remote possibility* of an unrecognized neoplasm in later life.

As far as hormones are concerned, I think hormones in the prepubertal child are risky, and, personally, I never use them. I will venture to say that most of the testes that come down with the use of hormones would come down anyway at the time of puberty, and I feel that it is an unjustified procedure to use either chorionic gonadotropins or androgens in young children.

One reason that there is controversy about what to do for undescended testicles is the difficulty in obtaining long term follow-up studies. Perhaps in future years we will have better statistics on the subject. For the time being, I think boys whose testicles never appear in the scrotum spontaneously and cannot be milked down into the scrotum manually should be operated upon in the preschool age. The testicles should be brought down with extreme care, without tension, and should be maintained in the scrotum, if at all possible, without any sort of a prosthesis, such as rubber bands, or other methods of traction.

Dr. Washington:

The surgical panelists agree that we should bring testes into the scrotum at the age 5 to 7. I am a little disappointed that they are not willing to boost the deadline up to the Boston level of 9 to 11. However, the surgeons who do these operations should act at the time they think they can do the best job.

I have seen boys of school age quite upset by the lack of one testicle. The use of hormones has been abandoned by everyone who has dealt with the problem recently, and there is evidence that damage may be done to the testicle by their use.

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# Sleep Disturbances in Young Children*

BETTY HUSE, M.D.†
MILDRED JANUARY, M.D.‡

Sleep disturbances in children, as in adults, may be caused by physical changes—illness, drugs, central nervous system lesions; by environmental conditions; or they may be "functional." This discussion is limited to "functional" disturbances of sleep in young children.

The word "functional," is used rather than "emotional" or "psychogenic," because all we can do is observe the behavior of an infant and see how he functions; we have no way of getting data from the infant as to his emotions or thoughts, if he has any.

This is a description given by observers of a newborn infant:[1]

> If we handled the patient roughly he cried weakly . . . and when we coddled him he showed contentment and settled down in our arms. When a finger was placed into his mouth he sucked vigorously. When he was held supine on extended hands and dropped two inches he would throw his arms out in fear, then flex them when he again came to rest. He would sleep after feeding and awaken when hungry, expressing his hunger by crying. His bowel and bladder function were normal.

The infant described by the observers, Nielsen and Sedgwick, was an anencephalic monster who on autopsy was found to have nothing in the central nervous system higher than the mesencephalon, not even thalami. Compare this description to Anna Freud's description of a normal newborn infant:[2] He "sleeps, wakes, cries, soon smiles, moves, feeds, empties his bladder and bowels."

Even at this extremely primitive level of mental development, then, sleeping is one of the basic functions; eating is another. So it is no surprise

* Presented as part of the course, Psychiatric Contributions to Pediatric Problems, given at University of Maryland School of Medicine, Baltimore, May 16–17, 1959.

† Teaching Analyst, Baltimore Psychoanalytic Institute.
‡ Assistant Clinical Professor of Psychiatry, Yale University.

that the first functional disturbances in a child are eating problems and sleeping problems. Furthermore, at the beginning of life, eating and sleeping are so closely tied together that they seem to be different sides of the same coin: The infant is hungry and awake, or he is not hungry and asleep.

Usually there is at least a short period in the physically healthy infant in which this state of affairs obtains. Then, at least in some infants, colic appears. This is often treated, especially by parents, as a feeding problem—"if only the right formula can be found it will be all right." Is it not also a sleeping problem? It certainly is to the parents! Little is really known about colic. After a while it wears off, seemingly without regard to any measures taken. Looking at an infant with colic, we could say that some basic need is not being met, but just what the need is and how to meet it baffles us.

After this disturbance there is usually a relatively quiet period of development until the baby grows to his next developmental crisis. This is what Anna Freud calls the change from "stomach love" to "love of mother." The young infant apparently functions well if his animal needs are met (such as feeding, firm gentle handling, protection from too intense external stimuli) and he plays with the person who cares for him as though that person were part of himself. Then, in the second half of the first year, he begins to perceive his mother as a separate person. Memory and other cortical functions develop. At this time, instead of needing only food, for example, he acts as though he also needs his mother—if he has his mother he will be cared for, without her he is lost. It is after this stage of development takes place that the first clearly defined sleep disturbances may be seen. Around 9 or 10 months of age, sometimes earlier, sometimes later, certain babies have difficulty in going to sleep and may wake once or more during the night, crying.

Such disturbances are sometimes reported as transitory, but sometimes instead of disappearing the difficulty in sleep increases. The child stays awake longer and longer, fussing, demanding, crying, calling mother back time and time again, finally sleeping in what seems to be sheer exhaustion—only to wake crying in the night at increasingly frequent intervals. At the same time, the child is fatigued during the day, eats poorly, and is irritable and temperish. The whole family becomes exhausted and angry at one another.

Usually the mother strenuously tries to meet the child's demands—she rocks, sings, gives extra food, returns again and again, shifts the bedtime and naptimes, allows the baby to play in the middle of the night, and tries everything she can think of. But this cannot go on indefinitely: not only the child but also the mother needs sleep. So the mother often gets the father to tend to some of the nightwaking in order to relieve her. This

makes things worse: the fear is loss of the mother, and when the father comes and not the mother the child's fear may be further increased. In desperation the mother may take a few days' vacation, but again the child's symptoms worsen, and the temporary rest for the mother is soon overbalanced by increased symptoms. If the child is sent to the hospital to determine what is wrong, again the problem is worsened.

As an illustrative case (and one which could no doubt be duplicated over and over), a father complained that his 16 month old daughter was "driving them crazy" by keeping herself and her parents awake most of the night. She had had some early feeding difficulties and colic, and always had been a light sleeper. When she was 15 months old her parents had gone away for a weekend, leaving her at her grandparents' home in another city. On their return, the marked difficulty in sleep appeared. It soon became too much to cope with. The father took over the nightwaking care, but as things got worse and worse the only thing he could think of to do was to sleep with the little girl on a studio couch so that at least he would not have to get up every time she awoke and screamed. By day she ate poorly, and was pale and very irritable.

Emergency advice to the father was aimed at convincing the child that she would not again lose her mother, and that her mother loved her and thought of her even when the child was asleep. To this end, the mother was to take entire care of the child at night and to continue to do so until the symptoms had entirely disappeared. The mother was to set up a bedtime routine that she could tolerate comfortably for whatever time was needed. At a regular bedtime, she was to give the little girl a bottle, preferably in the crib, and was to sit in a chair beside the crib without getting up until the child was firmly asleep. She could knit or read or otherwise occupy herself as she sat there. When she left she was to make quite a lot of noise to make sure that the child was sound asleep. She was advised to assure the child then, and also during the day, that she loved and thought of her even when she was asleep, and leave a piece of candy or some other small gift in the room for the child to find in the morning as a token of the truth of this. These parents, who were not only intelligent but quite willing to follow suggestions, adhered to this program. Within two weeks the child had ceased waking at night and went to sleep within 15 minutes after finishing her bottle.

Bobby was another child who had a sleep disturbance which was more complicated and of longer duration. His mother came to see me when he was about 3½ years old: for six months he had had a severe sleep disturbance. The mother was going to have a baby—her third—in about six weeks, and she planned to send him to his grandparents when she went into the hospital; he was to remain with them for two months. This was

considered an emergency situation, and, in the time available, an attempt was made to 1) make the separation at time of delivery as short as possible, and 2) try to alleviate the sleep symptoms before the next traumatic experience—the new baby.

This child's father and mother, both intelligent people, had wanted children as soon as possible after marriage, but were childless for 3½ years. The mother feared it was her fault and had almost given up hope when she became pregnant. The boy was a normal healthy baby, but did not do well on the breast and had to be weaned after two months, much to the mother's disappointment. Following this, there were many changes of formula. He was always a poor eater. He also had colic and was a poor sleeper. At about 8 months of age he screamed at bedtime, and his mother had to go upstairs and pat him every 15 minutes until he fell asleep exhausted. Information concerning the mode of onset of this trouble, as well as the length of time the disturbance lasted in its most severe form was not available, but apparently when it tapered off it left a child who would not be separated from a toy elephant which he called "Baby"; who insisted on having his father's attention whenever father was around; who apparently spurned his mother, was irritable, had many temper tantrums, and was very hard to manage.

He was precocious intellectually. He had a vocabulary of 20 words at 1 year of age, and when he was 2 his parents could talk to him as though he were a grown-up. A brother was born when he was 21 months old, at which time he was sent to his grandparents for two months. On his return he ignored the baby and was harder to handle than ever. Bowel training was accomplished with great difficulty, as was daytime dryness; he was still wetting at night when he was age 3. At some time his mother had thrown away his elephant, "Baby," when it had become very dirty, and he had then taken to carrying everywhere one of his father's handkerchiefs. He called it "Hanky," and took it to bed with him.

When he was 3 years old, his mother determined to train him not to wet the bed, moved him from a crib to a youth-bed, and took his "Hanky" away from him. It was at this time that severe sleep disturbance developed; this was accompanied by increased behavior disturbances during the day. Mother said, "There are 65 things he wants and all at the same time, and no one could possibly give him all the things he seems really to want." Because his appetite was so poor, tonsilloadenoidectomy was done four months later with no improvement.

The mother was seen six months later. The child's fear of losing her when asleep was explained and a regimen much like the one described for the previous case was outlined. During the first week of the regimen, the child would insist on the father putting him to bed, and the mother

would step aside; or the parents would be busy and the maid would put him to bed. The mother actually put him to bed only once during the week. The mother's notes for the day following the night she put him to bed illustrate very well his reaction: "Sunday: Very good all day. No nap and rather tired by night. We had guests for cocktails so maid put him to bed a little after 7. He woke at 9 hysterical. Wanted his father so I got him. Then he wanted a sip of ginger ale. Absolutely hysterical, unreasoning, threw himself around in bed, etc." On Monday: "Up since 5:30. Very worried about him. A tantrum at breakfast. Wanted to go out in the rain in the middle of breakfast. Sobbed when he couldn't. Cried for almost half an hour when his father went to work. Finally ate cold egg (with maid) but would not go to school. Fairly good off and on, but called for Daddy. Fell exhausted into bed for nap."

In desperation, and before intervention was possible, the mother went away for a few days' rest. On her next appointment it became clear why she had not been able to follow the suggestions: She believed that Bobby loved his father dearly but hated her, and she felt hopeless about being able to help him. She was convinced that she was no good as a mother. When she was able to understand that Bobby fought so fiercely with her just because he loved her so much, she said she felt she could stick to the bedtime regimen more strictly. Here is her note for the first determined effort to put him to bed: "He ate dinner with us—small tantrum over no chop for him. Father gave him his bone, he would not take mine. Then I bathed him, explained I was going to stay in his room, etc. Father came up to say goodnight. Wanted to know why Daddy wouldn't stay. Didn't want me. I settled down with book at 7:30. He got out of bed twice, then much conversation for ½ hour. First, he said he could not sleep with me in room; second, would I please stay there all night. What was Daddy doing—had he gone out? Was I going to sit with his brother, too? General conversation about events of day. Questions farther and farther apart. Asleep at 8:10. Left at 8:20. Got him up at 11. Woke at 4 whimpering, up at 6:30 for good—dry and cheery. Off to school." The following three nights were much the same, and he was reported as "angelic" in the daytime. At dinner "he ate like a bear."

Then twice the parents went out for the evening after he had been put to bed, and the following days he was "whiny, bad, and horrible." When this was pointed out to the mother, she was able to give up going out for the time being and felt the sacrifice well worth while when, within two weeks, Bobby was going to sleep promptly, sleeping all night, and no longer wetting his bed. She was able, now, to put him to bed, sit with him for a few minutes, and then leave him while he was still awake. With this improved relationship between them, she was able to discuss with him,

and to let him talk about, the new baby who was coming; and when she went to the hospital she spoke to him on the phone at least once a day. There was no exacerbation of his sleeping disturbance after the baby arrived.

Of course, not all this boy's problems were solved by this emergency treatment. Because of the severity of the disturbance and the emergency nature of the situation, it was necessary to treat at once the behavior to which he had regressed and treat him as though he were much younger. It was clear that it would be necessary later to unravel the problems that had been added and mixed with his early separation fear. Although his sleeping and eating problems and his bed-wetting disappeared and his general behavior improved, he still had a very hard time both at home and at school, and his character development had been significantly interfered with. He has continued to be in psychiatric treatment for some time since the time described here.

Needless to say, not all children with sleep disturbances as old as this boy should be handled in the same way. Under other circumstances it might have been better to start with more recent problems—such as his mother's pregnancy, his toilet training, his unusually strong attachment to his father, his feelings toward his brother. Had his disturbed course of development led him to a different solution, such as clinging to his mother at bedtime, or had his mother become involved in a pattern of overindulgence and overprotection toward him, it might have been very unwise to do anything to reinforce this.

There are very few cases of sleep disturbance in young children described in the literature, and in the cases reported the amount of detail varies. It has been possible to collect 15 cases: one analytically studied by Berta Bornstein;[3] five described more or less briefly by Fraiberg;[4] five by personal communication from one of us (M. J.); and four additional seen and studied in more or less detail by the other (B. H.).

Eight of the children were boys, seven were girls. Nine were first children, two were second children, one was a fourth child, and in 3 cases the sibling order was not specified. In five, the onset of severe sleeping disturbance was between 6 and 12 months. Nine started between the ages of 12 and 20 months. One started after 2 years of age.

Several factors were commonly present in those cases in which the information was sufficient to judge:

1) The mother had reason to fear that the baby would not live or be healthy. In 3 cases, several years of childless marriage had occurred in spite of continued efforts to have a child; in two, the previous pregnancy had ended with the death of a 6 or 7 month fetus in utero; in another, the patient was born with congenital heart disease, (the previous baby had been born at 6 months and died within a few hours, and there had

been a number of previous miscarriages); in two others, previous children in the family had severe illnesses. In the other 7 cases, information was not available on this point.

2) The child was very intelligent. Six of them had had IQ tests: one tested at 148, three at 150, one at over 179. The sixth (the second case presented) tested at 124, but the test results were probably too low because he would not cooperate well with the test procedures. In five others who had not been tested, the material shows definite intellectual precocity. In the other four there was insufficient evidence to judge.

3) As an infant, the patient was a poor sleeper and feeder. Ten of the children had both sleeping and eating problems in infancy. In one case it was stated that there were no feeding problems in infancy. In the others there was no mention of eating or sleeping during infancy.

4) There had been some sort of overnight separation between mother and child associated with onset of symptoms at an age when the child probably could not be adequately prepared for this experience. Such a separation had preceded the onset of symptoms in 12 of the children: in half of them the mother had gone to the hospital for such reasons as delivery of a baby, bleeding miscarriage, tuberculosis, and cancer of the uterus; in several the mother had gone away for the weekend; in one case the mother had had to devote herself to the care of an older child with tuberosclerosis when the patient was 8 to 14 months old, leaving the care of this child to the father and various relatives, and the child's sleep disturbance began when the mother returned to him.

These factors may, of course, not stand up when additional data are available since these are very few cases. They do, however, suggest the following possibility: it seems that a disturbed early infancy is a necessary foundation for the later development of a sleeping problem in early childhood. Whether this disturbed period is due to the way the very fearful mother behaves toward the infant, or whether a precocious cortical development of the infant in some way throws his development out of balance, or a combination of these factors, is not known. However, it seems that in this early period and in spite of heroic efforts, for some reason this infant has needs that cannot be met. On this foundation, the child develops to the period when he perceives his mother as a separate person, one whom he both loves and needs, and thus fears to lose. Then, in reality, the child loses his mother—for to the small child a single night can be an eternity. When the mother returns, the child's fear of losing her again is enormously increased. He fears losing her in sleep and struggles to stay awake. With this disturbance there is some regression to the earlier unsatisfactory period of life, with a resultant picture of a child who seems to want everything and is satisfied by nothing.

In a child who has had an early sleep disturbance, later stress periods

may reactivate the old problem, and the old and new problems may become entangled in complicated fashion. The second case reported is an example of this, and the case reported by Bornstein illustrates this very well.

It seems worthwhile to try to prevent such disturbances. Since the cause of a disturbed period in early infancy is so cloudy, it is difficult to see what preventive measures could be applied; perhaps help and support to a mother who has reason to fear for her baby might be useful. Given a disturbed early infancy, or at the first signs of sleep symptoms in the second half of the first year, it might be useful to explain to the mother the possible outcome of a separation from her child from the ages of about 6 months to 18 months. In a susceptible child, a break in routine may constitute a threat, so that some emphasis might be put on a steady relatively consistent bedtime routine for such a child. If the mother has to be away for an urgent reason, such as hospitalization, she could perhaps be prepared for the child's possible reaction upon her return; she could then be given some help as to how she might handle the child so that this reaction would not progress to a very distressing and disruptive full-blown sleep problem.

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# Sudden and Unexpected Deaths in Children

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The child who dies suddenly is a perennial subject for discussion in most children's hospitals throughout the country. The present survey is based on the examinations of autopsy protocols of children who came to this hospital and died within 24 hours or less during the three years, 1956–

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1958. All children in whom there was knowledge of pre-existing disease were eliminated from this survey. There remained the autopsy reports of 111 infants fulfilling the criteria of both sudden and unexpected death (table 1).

Tabulations of sudden deaths such as this continually serve a useful purpose since they help provide data for comparison with earlier data and, we hope, the answers to several important questions. Has there been any change in the etiological factors which cause sudden death in infants and children over the past few years as compared to those reported previously? What, if anything, can the medical profession do to lower the mortality of children to be admitted to the hospital in the future? Are there any new or better theories as to why these children die under circumstances which the pathologist and the clinician are at a loss to explain?

One fact that is apparent to everyone is that if a correct diagnosis is to be made, a good history is essential. It takes something of an expert to get the necessary detailed history from the parents of an infant who is brought to the hospital *in extremis* when the parents are so mentally disturbed that they are often incapable of giving an accurate history. This is complicated by the more than occasional guilt feelings that the parents have about what they did or failed to do. One report[1] written by those who are interested in the medico-legal aspects of this problem states, among other things, that if a police officer interrogates the parent, the subsequent medical history is not very likely to be correct. If, however, these parents are visited a few days later when things have quieted down and their mental state is somewhat more composed, the medical history may be very different and provide information of much help in making the diagnosis.

There has been a changing pattern in the causes of death at Children's Hospital. In 1945, Dr. Julius Loebl,[2] then a resident at this hospital, collected data which are quite comparable to those currently presented. One of the important causes of death he stressed in his report 14 years ago was diphtheria; yet in the present list, diphtheria is not mentioned. Loebl spoke of prematurity as a cause of death, a diagnosis which would not be very acceptable today; however, prematurity, even as a contributing factor, did not play a great part in the death of these children. He mentioned coagulation defects, of which the present group contains only one example (idiopathic thrombocytopenia). He devoted some time to accidental death which is not mentioned in the current list, since these patients are autopsied by the coroner. There is only one death in the present group (salicylate poisoning) which might be included under this heading. Thirteen per cent of Loebl's patients died of the Waterhouse-Friderichsen syndrome; there are only about half as many in the current statistics. Loebl's report concludes with a discussion on the thymus and status

TABLE 1

*Causes of Sudden Deaths in Children Admitted to Children's Hospital of D.C., 1956–58*

| No. of Children | Pathological Diagnosis | DOA | Incorrect Diagnosis | Correct Diagnosis |
|---|---|---|---|---|
| | **Infection** | | | |
| 6 | Septicemia | 3 | 1 | 2 |
| 6 | Waterhouse-Friderichsen syndrome | | | 6 |
| 2 | Meningococcemia | | | 2 |
| 4 | Acute laryngotracheobronchitis | | | 4 |
| 11 | Pneumonia: | | | |
| | 5 Broncho- | | 1 | 4 |
| | 1 Broncho- and lobar | | | 1 |
| | 5 Interstitial | 3 | 1 | 1 |
| 7 | Enteritis and gastroenteritis | 2 | 1 | 4 |
| 1 | Peritonitis | | | 1 |
| 2 | Hepatitis | | | 2 |
| 2 | Measles | | | 2 |
| 10 | Meningitis: | | | |
| | 4 *H. influenzae* | | 1 | 3 |
| | 5 *D. pneumoniae* | | | 5 |
| | 1 *L. monocytogenes* | | 1 | |
| 1 | Encephalitis | | | 1 |
| Total 52 | | | | |
| | **Heart Disease** | | | |
| 19 | Congenital heart disease | 5 | 4 | 10 |
| 5 | Congestive heart failure | | | 5 |
| 4 | Endocardial fibroelastosis | 1 | 2 | 1 |
| 2 | Myocarditis | 1 | 1 | |
| Total 30 | | | | |
| | **Hemorrhage** | | | |
| 4 | Intracranial | | | 4 |
| 3 | Pulmonary | | 2 | 1 |
| 1 | Adrenal and brain | | 1 | |
| 1 | Adrenal, lung and heart | | 1 | |
| 1 | Gastric | | | 1 |
| 1 | Idiopathic thrombocytopenia | | | 1 |
| Total 11 | | | | |
| | **Miscellaneous** | | | |
| 3 | Tracheoesophageal fistula | | | 3 |
| 5 | Aspiration gastric contents | 3 | | 2 |
| 1 | Volvulus | | | 1 |
| 1 | Perforation stomach | | 1 | |
| 1 | Dehydration and acidosis | | | |
| 1 | Adrenal cortical hyperplasia | | 1 | |
| 1 | Salicylate poisoning | | | 1 |
| 4 | Anemia, sickle-cell | 1 | | 3 |
| 1 | Acute glomerulonephritis | | | 1 |
| 1 | Leukemia | | 1 | |
| Total 18 | | | | |
| 111 | | 19 | 20 | 72 |

thymicolymphaticus—a factor which is rarely thought to be a cause of sudden death at present.

Surveys such as these have been approached in different ways. The medical examiner or coroner may present analyses with very different results from the present one because they are based largely on accident cases. Stowens,[3] writing from the Armed Forces Institute of Pathology, reported on the examination of the records of about 2,000 children from military institutions in different parts of the world; his results are also somewhat different from those of our survey. One would expect this to be so, since he was working with data and tissue collected from distant sources, and he did not have the opportunity for personal contact with the attending physicians. A recent report[4] from St. Christopher's Hospital in Philadelphia, an institution similar to Children's Hospital of the District of Columbia, presents results which are quite comparable to our own.

In the present group 70 per cent of the patients were under 1 year of age, 25 per cent were under 1 month and 15 per cent were under 1 week. The death of the largest number of children (52 of the 111, or 47 per cent) could be ascribed to infection. The first category in this group is septicemia. The pathologist is often in a quandary to be certain that a given patient really died of septicemia if the diagnosis is based only on a positive postmortem blood culture and some tissue evidence of infection. Actually the clinical diagnosis may be more reliable. The same comments apply to the pathological diagnosis of pneumonia, particularly when a diagnosis of interstitial pneumonia is made. Here we are probably treading on rather unsafe ground by saying that this was the cause of a child's death since pathological evidence of interstitial pneumonia is found so frequently.

The Waterhouse-Friderichsen syndrome is apparently a condition we will always have with us; we saw it in 1945 and we continue to find it. While the mortality rate for meningitis has dropped sharply, it continues to be a significant cause of death. Of the 10 deaths from meningitis in this series, four were due to *Hemophilus influenzae*, five were due to *Diplococcus pneumoniae*, and one was due to *Listeria monocytogenes*. The last-mentioned organism has not appeared in any previous list of deaths such as this at this hospital. It is an organism which has always been with us, but rather recently hospital bacteriologists have realized that some of these organisms which previously were called contaminants are actually pathogens. *L. monocytogenes* is an organism which will respond to antibiotics; therefore, if an early diagnosis can be made and treatment instituted promptly the patient's life can frequently be saved.

Abnormalities of the heart account for 27 per cent of the total deaths, and in this group congenital heart disease stands out as the most common single cause of death; this is also the most commonly missed diagnosis

(see table 1). It is encouraging to note that one of the four cases of endocardial fibroelastosis was diagnosed during life, as this diagnosis was not made 15 years ago except at necropsy.

Eleven deaths were due to hemorrhage. Those due to pulmonary hemorrhage were usually diagnosed as pneumonia, an almost impossible differential diagnosis to make as the clinical findings are very much alike and there is insufficient time for one to make any fine differentiation in these children who die so quickly. Two children died of unrecognized renal hemorrhage; the exact cause of such hemorrhage is not always known but in most cases is presumed to be due to infection.

There were three patients with tracheoesophageal fistulae, all of whom were diagnosed correctly before death and all of whom died within 24 hours after admission. The factors initially responsible for death lie elsewhere. If these infants are to be salvaged, the physician who sees them when they are born must be alert to the situation and make his diagnosis early.

One infant died of perforation of the stomach. This 4 day old infant was thought to have intestinal obstruction and a rubber tube was passed into the stomach. Whether this tube actually caused the perforation we do not know. This is, however, a possibility, and we have had evidence in the past that tubing may cause perforation of the esophagus or stomach on occasion.

One child who died of adrenocortical hyperplasia with complicating dehydration and acidosis and presumably electrolyte imbalance might have been saved if more time had been available.

There were four patients with sickle-cell anemia who died. Two of these also had bronchopneumonia, and one had erythropoietic aplasia of the bone marrow. It is difficult to state exactly of what these children died, but basically these deaths are probably attributable to the fact that these children had sickle-cell anemia and were good subjects for bronchopneumonia.

In the past, both at Children's Hospital and in other large medical centers, only sporadic attempts have been made toward looking for a virus as the responsible infecting organism. The results so far have been very disappointing; in only a small percentage has a virus been recovered. This should change as more and more hospitals develop virus laboratories such as that which now exists in this hospital. Undoubtedly, increasing attempts will be made with increasing success to isolate a virus from these children who die suddenly. It will be difficult to determine whether or not the virus isolated is the etiological factor contributing to the death of the patient.

In table 2, a more detailed presentation of the 20 children in whom the clinical diagnosis differed from the pathological diagnosis is provided. This would appear to be a rather large number until it is noted that eight

TABLE 2

*Children in Whom the Clinical Diagnosis Differed from the Pathological Diagnosis*

| Age | Time in Hospital | Clinical Diagnosis | Pathological Diagnosis |
|---|---|---|---|
| 8 mo. | 1 day | Bronchopneumonia | Congenital heart disease |
| 23 hr. | 1½ hr. | Tracheoesophageal fistula | Congenital heart disease |
| 5 wk. | 7½ hr. | Bronchopneumonia | Congenital heart disease |
| 9 mo. | 5 hr. | Septicemia—Meningitis | Congenital heart disease |
| 9 mo. | 1½ hr. | Pneumonia | Endocardial fibroelastosis—Pulmonary edema |
| 7 mo. | 13 hr. | Congenital heart disease | Endocardial fibroelastosis |
| 1½ yr. | 10 min. | Pneumonia—Possible meningitis | Acute myocarditis—Septicemia |
| 6 da. | 12 hr. | Bronchopneumonia | Pulmonary hemorrhage—Renal infarction |
| 12 da. | 2 hr. 40 min. | Bronchopneumonia | Pulmonary hemorrhage |
| 9 mo. | 9 hr. | Severe gastroenteritis—Dehydration—Hypernatremia | Hemorrhage of the adrenals, lungs, heart—Focal interstitial pneumonitis |
| 5 yr. | 1 da. | Hypoglycemia—Infection | Bronchopneumonia—Focal brain hemorrhage—Adrenal hemorrhage—Cerebral edema |
| 18 mo. | 1 hr. | Tumor, right lower quadrant | Acute ulcerative enteritis—Multiple bacterial emboli—Hepatitis |
| 8 wk. | 19 hr. | Intestinal obstruction—Pneumonia | Septicemia with pulmonary abscess—Enteritis |
| 3 mo. | 40 min. | Acute gastroenteritis—Dehydration | Interstitial pneumonitis—Dehydration |
| 7 mo. | 15 min. | Acute adrenal insufficiency | Bronchopneumonia—Esophagitis |
| 2 wk. | 11 hr. | Bronchopneumonia | *H. influenzae* meningitis |
| 13 da. | 13 hr. | Starvation—Dehydration | Meningitis, due to *L. monocytogenes*—Encephalitis—Necrosis of the adrenal glands |
| 4 mo. | 15 min. | Meningococcemia | Acute leukemia |
| 4 da. | 1 da. | Intestinal obstruction | Perforation of the stomach |
| 18 da. | 1½ hr. | Feeding problem | Adrenocortical hyperplasia—Dehydration—Patent ductus arteriosus |

of these patients were in the hospital less than two hours. Heart disease was the most common disease missed, accounting for seven deaths. Most of these children had a complicating pneumonia which was either a direct or contributing cause of death.

There is much variability in the reports of others who have made similar surveys. Stowens[3] has stated that in 82 per cent of the cases of sudden death, the autopsy tissue which was submitted to the Armed Forces Institute of Pathology showed no recognizable pathological entity. Part of this puzzling circumstance is undoubtedly due to the fact that he was working under the disadvantage of never personally seeing any of these patients or having any direct contact with the attending physicians. In a recent issue of *Lancet*[5] the statement was made that the cause of sudden death in children is largely unknown. Werne and Garrow[6] mention deaths where the baby is found dead in bed. These deaths, most of which occur during the first few months of life, are believed by them to be due to infection. They mention a series of infants found dead in which 90 per cent had gross or microscopic evidence of mastoiditis. This is a most unusual finding. They also noted that fulminating respiratory disease is a common cause of sudden, unexpected and apparently unexplainable death. It is important to remember that such infections are not necessarily the cause of death.

While we have assigned a cause of death to each child in the present survey, it is obvious that there were a good many of these that cannot be satisfactorily explained. Most of the theories at the present time attempt to explain these by a sudden reflex disorder involving the autonomic nervous system. Handforth[7] has suggested the possibility of laryngeal spasm; he was able to cause death in animals by cutting off their air supply for a period of a relatively few seconds; at autopsy the changes in the lungs were similar to those seen at autopsy in some children who die unexpectedly and suddenly.

What can we do to reduce this mortality? For one thing, those physicians who see newborn babies in respiratory distress can think of tracheoesophageal fistula, make the diagnosis early, and refer them to the operating surgeon as soon as possible instead of waiting for as long as three days when the infant is almost sure to have aspiration pneumonia. While we will probably continue to lose infants with laryngotracheobronchitis and Waterhouse-Friderichsen syndrome, we might be able to do better if when we first see the patient we keep these diagnostic possibilities in mind and institute treatment immediately.

In conclusion it may be said that infection is still the most common cause of sudden or unexpected death in children; the role of viruses as a causative factor has not definitely been proved, but it is probable that they

will become increasingly important. Some deaths due to infection or congenital anomalies can be prevented if recognized promptly, and specific treatment given; many of these deaths cannot be explained on an anatomic basis, and some are certainly functional in origin. A carefully performed autopsy continues to be of great value in determining more definitely the cause of sudden death in infants and children.

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# The Editor's Column

## EVOLUTION IN MEDICAL EDUCATION

A recent evaluation of the teaching program in the medical department of Children's Hospital of the District of Columbia has forced the medical staff to focus on teaching in a hospital that is committed to education and research. The uniqueness of medical education requires special orientation. Here the physician must deal with theoretical as well as practical knowledge of medicine, and apply this knowledge in a logical and humane fashion to people. In the past century medical education has undergone revolutionary changes which have resulted in the highly integrated and specialized medical centers which are the nuclei for student and graduate education. These educational centers, in part, are staffed by university or hospital academic personnel. Children's Hospital of the District of Columbia is undergoing a similar evolution in its teaching and training program. The complexities of modern medical care and research have made this mandatory in order to assure its future as a service and educational institution.

The three requisites for a teaching hospital are present in part or in their entirety at this hospital. The first is sufficient patient population for teaching purposes. The second is students who have the desire for knowledge, and the third is the unequivocal desire of the institution to commit itself fully to a teaching and research program.

What role does a teaching hospital play in medical education? This hospital is responsible for teaching at three levels. First, to teach medical students; second, to train physicians in the specialty of pediatrics; and third, to conduct a continuous postgraduate program for the pediatricians of the community. There is no doubt that the quality of medical care in any given community is directly related to the quality of this continuous postgraduate education. Only in this manner can the public's demand for superior medical care expected of a specialist in pediatrics be rendered. The graduate training program must be of such quality that it attracts a house staff of high caliber and attracts the pediatric community to its major teaching exercises. This requires a considerable investment of time in its preparation as well as a vigorous creative approach. This program then is the responsibility of those who by interest and by training participate as teachers.

Are full time teachers necessary? The evolution of medical education indicates that this is a necessity; it is an essential part of medical education. Those institutes without a hard core of full time academic staff suffer in their educational program and therefore are unable to attract an adequate house staff. There must be an adequate balance between those who are professional educators and practice hospital medicine and those who are interested in teaching but whose professional interest is private practice. One group cannot do without the other. Furthermore, the compounding of medical knowledge has increased at such a rapid rate that an up-to-date teaching program requires men who spend their time keeping abreast in a certain segment of medical care or specialization. The rate of medical advance is so rapid that even those in special fields find it difficult to keep abreast. The full time academic staff, each member of which, by his training and research in his specialty, is able to gather more experience with the complex disorders of children. Therefore, other physicians, whether at the undergraduate or graduate level, are able to benefit by their experience in hospital pediatrics.

Do men in practice play a role in a teaching hospital? The answer to this is an unequivocal "yes." It is clear that men in practice have a much wider range of experience in the care of the variety of problems that one may not see in the hospital. Since we are training physicians who will go out into practice, this aspect of pediatrics must be incorporated in any

well rounded training program. It may be that a certain physician is uncomfortable in teaching, for example, as an attending physician on the ward, although he may want the service for what he may learn from it. On the other hand, he may be extremely valuable as a teacher in the outpatient department. It is generally agreed that outpatient medicine should receive major emphasis for those trainees who expect to go into practice. In order to obtain the depth of training and teaching necessary in various aspects of the training program, it may be advisable to break down the teaching teams into those who, by training and knowledge, are best suited for ward teaching and those who are best suited for outpatient teaching.

As this hospital increases its reputation as a referral center resulting from its teaching and research programs, the complexities of the diagnostic and therapeutic problems will increase many fold. Therefore, the hospital professional staff must be ready to deal with the increasing demand for more complex services. This applies not only to the medical services, but also to surgery and its subspecialties as well as x-ray, pathology, and psychiatry. Since the teaching and research done by the professional staff basically determines the medical stature of an institution, the degree and spirit with which the academic program is pursued will determine its local as well as its national stature.

FELIX P. HEALD, M.D.

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# Book Reviews

*The Placenta and Fetal Membranes.* Edited by CLAUDE A. VILLEE, Ph.D., 404 pages, 87 figures, Baltimore: The Williams and Wilkins Company, 1960, $10.00.

In four sections, this book represents for the pediatrician a source reference on the role of the placenta. In Part I there are eight reviews, two of which might be of special interest to the pediatrician—transmission of antibodies from maternal circulation to the fetus, and placental function and fetal nutrition. These sections, plus portions of Part III (review of the literature) might well make this text the best available stepping-off point for one interested in research on the role of the placenta in newborn welfare. For example, Brambell and Henning's section on transmission of antibodies is far more a review of unsolved questions than it is a recitation

of facts. The literature review is available to the reader who will go further. A welcome bonus of this section is the presence of pithy summaries on each reference cited.

The other two parts of the book are Part II, Proceedings of a Conference held in November 1958 under the sponsorship of the Association for Aid to Crippled Children on placenta research and research needs, and Part IV, a list of investigations in the field. The proceedings of the conference reflect a very basic approach concerned primarily with the morphology, physiology and biochemistry of the placenta.

ROBERT H. PARROTT, M.D.

*Notes on Infant Feeding.* By STANLEY GRAHAM, LL.D., M.D., and ROBERT A. SHANKS, M.D., fifth edition, 76 pages; Edinburgh and London; E. & S. Livingstone Ltd., 1960. (The Williams and Wilkins Co., Baltimore, exclusive U. S. agents) $2.00.

This book is written primarily for the use of medical students at the Royal Hospital for Sick Children in Glasgow. It makes delightful reading, but physicians in this country will find in it the aura of another age. We learn the value of rose-hip syrup and the problems of using open end boat-shaped bottles. Liquid milk must be boiled for three minutes as soon as it is brought into the home. We are dismayed to find directions for preparing oat-flour porridge and seived vegetables with bone.

The accepted methods for feeding infants in Scotland vary considerably from those we have become accustomed to during the past decades in the United States. Some of these differences are based on the availability of certain food products, and other differences are a matter of custom which cannot at the present time be considered inferior or superior to our own customs. For example, more evidence is necessary before we can be certain that it is better to take a few days rather than two weeks to establish full caloric feedings after birth. Both countries produce thriving babies, and it is well to remember, as the authors point out, that rapid gain in weight is not the only criterion for good health in young infants.

Formula making seems unnecessarily complicated in Scotland, although the authors note with approval the trend to simplify the subject and avoid the old "percentage feeding." Caloric requirements are calculated on expected weight with various mathematical adjustments being made for the infant who is severely undernourished. Half-cream dried milk without extra dilution is used routinely in the first two to three months of life, and then full-cream dried milk. One teaspoonful of sugar is added to each bottle replacing one ounce of the calculated amount of milk. The first solid food is started when the infant is not satisfied with 30 ounces of formula, but this seems to occur rather late. As a rule, porridge is begun at three to

four months, sieved vegetables at four to five months. No mention is made of meat, except for fish, in the first year of life. The variety of the diet seems rather meager by our standards, but while variety provides a safety factor, the lack of it does not necessarily mean poor nutrition.

Besides acceptable differences in custom, there are certain statements of the authors which I do not think hold true in this country. "The most frequent cause of failure to thrive is underfeeding . . . Homogenized vegetables in tins may be used when fresh foods are not available . . . Ascorbic acid in tablet form is more expensive (than orange juice)." I also question the validity of the arguments presented in the book for continuing the same number of calories (i.e., 45) per pound per day throughout the first year of life. Certainly we find empirically that our infants eat relatively more in the first six months of life than in the second six months, and this finding is in accord with the recommendations of the Food and Nutrition Board of the National Research Council and the Academy of Pediatrics. Interestingly enough, whether it is cause or effect, the weight gain of infants in Scotland conforms to this caloric intake and remains approximately the same (and rather low) per month throughout the first year of life.

The authors should certainly be commended for emphasizing the importance of breast feeding, although most of us would question the necessity of breast-milk banks for prematures. They also point out the danger of giving too much vitamin D when milks as well as proprietary cereals are fortified, and then cod liver oil is added.

After reviewing two previous editions of this book, I find that trends in infant feeding in Scotland are much like those in this country, primarily toward simpler formulae and earlier feeding of solid foods. We have certainly gone faster and further in these trends, perhaps too far in some respects. This volume should encourage us to review our habits and avoid extremes.

This opportunity for critical reappraisal of our own practices is the only value of this book for pediatricians in the United States; our medical students would only be confused by reading it. Despite the convenient pocket-sized form of this text, the 23 pages in Nelson's *Pediatrics* are more complete and appropriate for us.

MARGARET F. GUTELIUS, M.D.

*Communicable and Infectious Diseases.* By FRANKLIN H. TOP, M.D. and collaborators, edition 4, 812 pages, 122 figures and 15 color plates, St. Louis: The C. V. Mosby Company, 1960, $20.00.

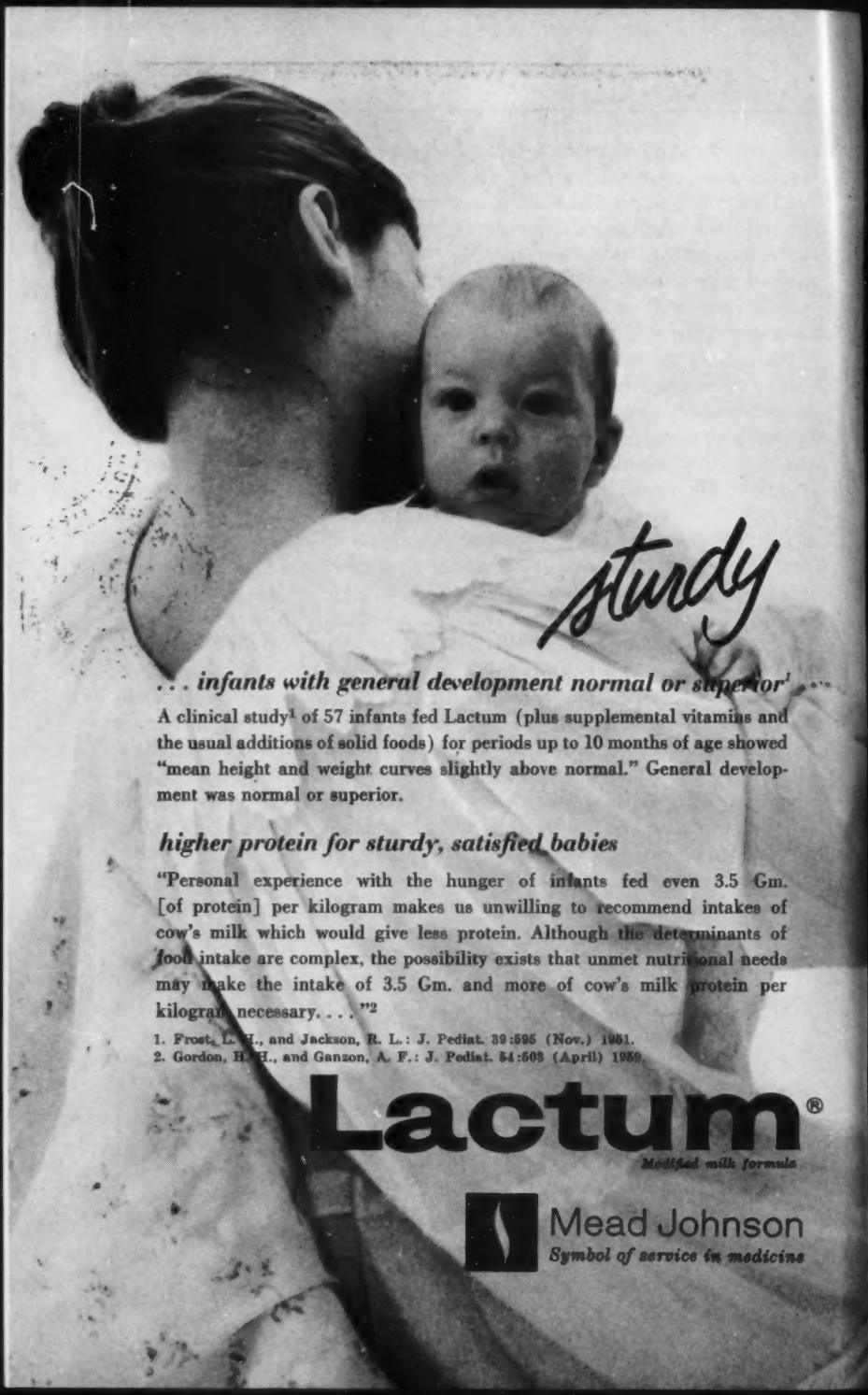
This fourth edition pleads well for the rational approach to infectious diseases. This is most evident in the introductory chapters on "general

considerations" but is preserved in most of the remaining chapters on specific diseases or syndromes. At the same time, descriptive clinical features are well presented. The pediatrician, however, may find that the approach is not directed strongly enough toward infants and children. Despite the fact that most of the illustrations depict children and much of the subject matter concerns children, few of the collaborating infectious disease consultants are primarily pediatricians, and some of the chapters fail to consider in sufficient detail either clinical or therapeutic matters important in the care of children. For example, little or no attention is given to infectious croup as a syndrome; treatment discussion (as in discussing the use of chloramphenicol in *H. influenzae pneumoniae*) fails at times to consider dosage by weight; no clinical description is given of herpangina and herpes simplex gingivostomatitis, common and sometimes perplexing infectious diseases of children; data in tables on the incidence of acute respiratory disease all refer to adults.

One of the universally applicable messages of the text comes from Dr. Lepper in his chapter on chemotherapeutic and antibiotic agents. After reviewing concisely the chemistry and mechanism of action of antibacterial agents, he presents a table on usefulness of sensitivity tests for various organisms with different drugs. In the table, N. I. means "Not indicated." N. I. is the most common entry, graphically bringing home the point that some underlying knowledge of both organism and antibiotics might help toward improving antibiotic usage by dispelling the frequently wasteful, automatic "culture *and sensitivities*" order. Also useful in this chapter are tables of minimal inhibitory concentrations of different antibiotic agents for different bacteria, combined with distribution of serum levels likely to be obtained with varying dosages and routes of administration

Summing Up: Rational, yet clinically practical; a bit too adult.

ROBERT H. PARROTT, M.D.

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1. Frost, L. H., and Jackson, R. L.: J. Pediat. 39:595 (Nov.) 1951.
2. Gordon, H. H., and Ganzon, A. F.: J. Pediat. 54:603 (April) 1959.

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